STUDY OF RHYTHM DISTURBANCES IN ACUTE MYOCARDIAL INFARCTION IN GOVERNMENT ROYAPETTAH HOSPITAL

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CERTIFICATE

This is to certify that this dissertation entitled **"STUDY OF RHYTHM DISTURBANCES IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION IN GOVERNMENT ROYAPETTAH HOSPITAL"** is bonafide work done by **Dr. S. SUDHA** postgraduate student, department of internal medicine , KMC Chennai -10 under my direct guidance and supervision in fulfillment of regulations of the Tamilnadu DR. M.G.R Medical university for the award of M.D Degree Branch I ,part II (general medicine) during the academic period from may 2005 to march 2008.

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CONTENTS

INTRODUCTION

Despite impressive strides in diagnosis and management of acute myocardial infarction in the past three decades, acute myocardial infarction continues to be a major health problem. Projection from World Health Federation is that the burden of ischaemic heart disease in the developing countries will become similar to that of developed countries.(1) About 50% of death from acute myocardial infarction occur within 1hr of the event and are attributable to arrhythmias most often ventricular fibrillation.

Ischaemic injury can produce conduction blocks at any level of the atrioventricular or Intraventricular conduction systems. Such conduction block can occur in the atrioventricular node producing various grades of AV block. Conduction block can occur in either main bundle branch producing right or left bundle branch block or in the anterior and posterior fascicle of left bundle branch, producing left anterior &left posterior fascicular blocks respectively.

AIM OF THE STUDY

- To study the incidence of rhythm disturbances in acute myocardial infarction
- To evaluate the age, sex distribution & various other risk factors in relation to rhythm disturbances occurring in acute myocardial infarction
- To correlate between the different types of rhythm disturbances in relation to the location & type of acute myocardial infarction
- To evaluate the incidence of rhythm disturbances in thrombolysed & non thrombolysed patients

REVIEW OF LITERATURE

CARDIAC CONDUCTION SYSTEM

The structures that make up the cardiac conduction system are the SA node, Internodal pathway, the AV node, the bundle of HIS and its branches and the purkinje system. The SA node is the normal pacemaker. Impulses generated in SA node pass through the atrial pathway to the AV node through this node of the bundle of HIS and through the branches of bundle of HIS via the purkinje system to the Ventricles.

I) ANATOMY

The SA node is located in the right atrium near the anterior border of the opening of superior vena cava. (2) The AV node lies near the orifice of the coronary sinus in the annular and septal fibers of the right atrium.(2) The three bundles of right atrium that conduct impulses from SA node to AV node are the anterior Internodal tract of BACHMAN, the middle Internodal tract of WENKEBACH ,the posterior Internodal tract of THOREL.

From the AV node the AV bundle, passes forward in the lower part of membranous septum and divides into right and left fascicle. These run down in the right and left Ventricle, one on either side of the

ventricular septum, covered by endocardium. In the lower part of the ventricles, they break up into numerous strands which end in the papillary muscles in the Ventricle (2)

BLOOD SUPPLY

The vascular supply of the SA node is via a small artery that arises about two cm from the ostium of the right coronary artery in approximately 60% of the population and from proximal part of the left circumflex artery in 40% of the population.(3)

In 90%of population the blood supply of AV node is via the AV nodal branch of the dominant right coronary artery. In the remaining 10%, the blood supply is via a branch of the dominant circumflex coronary artery.

The bundle of HIS and the interventricular septum have a dual blood supply ,which arise from the posterior descending branch of right coronary artery and septal perforating branch of left anterior descending artery.

The right bundle branch and left posterior division of the left bundle branch have a dual blood supply from left anterior descending and right coronary arteries, while left anterior division is supplied by septal perforators originating from left anterior descending coronary artery.

NERVE SUPPLY

The SA node develop from structures of the right side of embryo and AV node from left, that is why in adults, the right vagus is distributed mainly to SA node and left vagus to AV node. Similarly the sympathetic innervation of right side is distributed primarily to SA node and of left side is distributed to AV node.

II) SPREAD OF CARDIAC EXCITATION (4)

Depolarization initiated in SA node spreads radially through the atrium, and then converges on the AV node. Atrial depolarization is complete in 0.1 sec. Because of conduction in AV node is slow, there is a delay of 0.1 sec (AV nodal delay) before excitation spreads to Ventricles .From the top of the septum ,the wave of depolarization spreads in the rapidly conducting purkinje fibers to all parts of ventricle in 0.08 to 0.1 sec. In humans, depolarization of ventricular muscle starts at the left side of the interventricular septum and moves first to the right across the mid portion of the septum. The wave of depolarization then spreads down the septum to the apex of the heart .It proceeds from endocardial surface to epicardial surface. The parts of the heart to be depolarized last are the posterior basal portion of the left ventricle, the pulmonary conus and the upper most portion of the interventricular septum.

SPEED OF CONDUCTION

Tissue	Conduction Rate (m/s)
SA Node	0.05
Atrial Pathway	1
AV Node	0.05
Bundle of HIS	1
Purkinje System	4
Ventricular Muscle	1

III) ACUTE MYOCARDIAL INFARCTION

Criteria for diagnosis of acute evolving or recent Myocardial Infarction (5)

Either one of the following criteria satisfies the diagnosis of acute, evolving or recent myocardial Infarction

1. Typical rise & gradual fall (troponin) or more rapid rise & fall (CPK MB) of biochemical markers of myocardial necrosis with at least one of the following

   a) Ischaemic symptoms

   b) Q waves in ECG

   c) ST segment depression or elevation

   d) Coronary artery intervention (e.g., coronary angioplasty)

OR

2. Pathological findings of acute MI

Criteria for established Myocardial Infarction

Either of the following criteria satisfies the diagnosis for established Myocardial Infarction:

1. Development of new pathological Q waves on serial ECG readings. The patient may or may not remember previous symptoms.

2. Pathological findings of a healed or healing Myocardial Infarction.

IV) RISK FACTORS

- **Unmodifiable Risk Factors**

a) Age and sex

Obviously these risk factors cannot be corrected; however there is considerable evidence that hormone replacement therapy may reduce the risk of heart disease in post menopausal women.

b) Family History

Coronary Artery Disease (CAD) runs in families. This may be due to genetic factors or the effect of shared environment. At present it is estimated that about 40%of risk is controlled by genetic factors & 60% by environmental factors.

Modifiable Risk Factors

The absolute risk for development of CAD over the next decade can be estimated for men &women by FRAMINGHAM RISK Tables (6).

a) Smoking

A strong dose response relationship between cigarette smoking and CHD has been observed in both sexes, in the young, in the elderly and in all racial groups (7). There is no evidence that filters or other modification of cigarettes reduces the risk (8).Exposure to environmental tobacco smoke or passive smoking has been recognized as a modifiable risk factor (9).Exposure to tobacco smoke by non smokers was consistently associated with a 20 to 30% increase in risk. The increased risk in smokers is due to their increased vascular reactivity. Smokers have increased levels of fibrinogen and increased platelet aggregrability.

b) Hypertension

Several major prospective epidemiological studies have found that both systolic and diastolic hypertension have a strong relationship to CHD (10). A metaanalysis of 17 randomized trials of antihypertensive drugs in patients with mild to moderate Hypertension has found that the risk of CHD was lowered by 10 %. Blood pressure can be lowered by weight loss, exercise, salt restriction, avoidance of alcohol and drugs.

c) Left Ventricular Hypertrophy

It is defined by either electrocardiogram or echocardiography. It is an important independent risk factor roughly doubling the risk for cardiovascular death in both men and women (11). Left ventricular hypertrophy is associated with obesity, excessive salt intake, advanced age and heredity (12).Several studies have found that Angiotensin converting enzyme inhibitor reduces left ventricular mass by 12%, calcium channel blockers by 11%, beta blockers by 5% and diuretics by 8%.

d) Hyperlipidemia

The risk for CHD is positively correlated with total serum cholesterol concentration (13), which in turn is highly correlated with Low Density Lipoprotein (LDL). The risk is inversely related to plasma

High Density Lipoprotein (HDL) cholesterol concentration. LDL lowering can be accomplished by pharmacological and non pharmacological interventions. Achieving a desirable body weight will reduce LDL cholesterol level in most overweight individuals (14).Among cholesterol lowering drugs, Statins are found to be highly useful.

e) Lipoprotein(a) – Lp(a)

It consists of an LDL particle linked via a disulphide bond to an Apo lipoprotein (a) of polypeptide chain. Because of homology between Lp (a) and plasminogen, it serves as a competitive inhibitor for plasminogen binding and thus may inhibit fibrinolysis. Nicotinic acid and estrogen appear to lower Lp (a) levels.

f) Diabetes Mellitus

It is an independent risk factor for CHD, increasing risk by 2-4 times (15). Diabetic patient without history of MI have a high risk of coronary mortality as non- diabetic patients with a history of MI (16). Diabetic women have twice the risk of recurrent MI as compared to diabetic men (17).Pharmacological interventions to control dyslipidemia and Hypertension has been shown to have a clearly favorable effect on CHD events in type2 Diabetes Mellitus.

g) Metabolic Syndrome

Metabolic Syndrome a condition characterized by metabolic risk factors and increased risk of coronary heart disease. The Metabolic Syndrome can be said to be present, when a person has three of the following:

1. Abdominal Obesity (waist circumference >102cm in males,> 88cm in females)
2. Triglycerides >150mg/dl
3. HDL>40mg/dl in males and >50 mg/dl in females
4. BP >130/85mmHG
5. Fasting Glucose > 110mg/dl

h) Physical inactivity

It is an independent risk factor for CHD and roughly doubles the risk (18). There is a dose response relationship between the amount of exercise performed weekly and death from cardiovascular disease and all causes. Physical inactivity slows the progression of atherosclerosis in humans.

i) Obesity

Obesity is defined as Body Mass Index ≥ 30, is a major risk factor for CAD (19). It is associated with insulin resistance, hyperinsulinemia, Type2DM, systemic hypertension, low HDL, hypertriglyceridemia, inflammation, thrombosis, diastolic dysfunction and left ventricular hypertrophy (20).

j) Homocysteine

Hyperhomocysteinemia is an independent risk factor for CAD (21). A series of studies have found a linear dose response relationship between plasma homocysteine levels and a mortality rate of 4.5% for patients with higher levels of homocysteine. Nutritional supplementation with folic acid can lower homocysteine levels in many individuals.

k) Alcohol

Mild to moderate alcohol consumption (2-4 units/day) is associated with reduced rates of CHD (22). However heavy drinking is associated with hypertension and excess cardiac events.

l) Infection/ Inflammation

Recent publications have furnished evidence in support of a role for Chlamydia pneumonia, CMV, or other infectious agents in atherosclerosis. There is evidence that markers of inflammation

correlate with coronary risk. For example, elevated plasma C reactive protein can prospectively predict risk of MI and correlate with outcome of patients.

m) Psychological factors

The role of personality, environment, social support, social contact, stress, depression has all been associated with increased risk for Coronary Heart Disease.

V. ECG CHANGES

1) Hyper acute phase

The characteristic changes in the leads that are oriented to the infarcted surface are as follows (23).

- Increased ventricular activation time
- Increased amplitude of R wave
- Slope elevation of ST segment
- Tall and widened T waves

2) Fully evolved phase

- The myocardial necrosis is represented by QS complex
- Injury is represented by elevated and coved ST segment

- Ischaemia is represented by pointed, inverted , symmetrical T waves

3) Following the fully evolved phase of MI, there is graded resolution of abnormalities. The elevated ST segment gradually returns to baseline, becoming predominantly isometric, once again the inverted T wave gradually regains positivity.

VI. LOCALISATION OF MYOCARDIAL INFACTION

1) Anterior Wall MI

a) Extensive anterior wall MI: typical changes in leads I, aVL, and precordial leads.

b) Anterosptal wall MI: Typical changes occur in leads V1 to V4.

c) Anterolateral wall MI : Changes occur in LI, aVL, V4 – V6.

2) Inferior wall MI

Typical infarction pattern in lead II, III, aVF with reciprocal ST segment depression in right precordial leads.

3) Inferolateral wall MI

Typical pattern seen in leads II, III, aVF, V5 and V6.

4) Posterior wall MI

Diagnosed by inverse or mirror image changes which will be detected by electrodes oriented to the uninjured anterior wall.

- Tall and slightly widened R wave
- Depressed and concave upward ST segment
- Upright, widened and tall T wave.

5) Right ventricular infarction

Typical pattern in V1, V4R, V5R, V6R. Of these V4R is the most sensitive (24).

There is elevation of ST segment inV1 and ST segment depression in V2: discordance relationship (25).

6) Infarction of Atria

The following features are seen

- Elevation of PR segment in clinical setting of MI
- Abnormalities of P wave, 'P' wave can become widened, notched or slurred.
- Abnormalities of atrial rhythm
- There may be associated SA block.

VII. CLINICAL FEATURES OF ACUTE MI

A) Symptoms

1. Pain

Most patients present with chest pain. The pain is similar to angina in location and radiation, but is more severe. It may radiate to neck jaw, shoulders, back, or one or both arms. Nitroglycerine has little effect. Infarction is common in early morning. Early morning hours are associated with raise in plasma catecholamine and increase in platelet aggregrability (26).

2. Other symptoms

Indigestion or heart burn; nausea and or vomiting associated with chest discomfort.

Persistent shortness of breath

Weakness, dizziness, lightheadedness, loss of consciousness

3. Painless Infarction

Common in elderly and diabetic patients.

B) Signs

1. General

Patients appear anxious and are often sweating profusely. There may be bradycardia or tachycardia. Blood pressure may be high or low in patients with shock.

2. Chest

A clear lung field is a good prognostic sign. More extensive rales suggest pulmonary edema.

3. JVP

Jugular venous distension indicates right ventricular infarction.

4. Heart

Soft heart sounds indicate LV dysfunction. Atrial and ventricular gallops may be present. Mitral regurgitation murmur indicates papillary muscle dysfunction. Pericardial friction rub is common in large transmural infarction (27).

Markers of Myocardial injury

The markers of myocardial injury are cardiac troponin T or I, creatine kinase MB isoenzyme (CK-MB).

The characteristics patterns of elevation of biomarkers are as follows:

Biomarkers	Time of initial elevation	Time of peak elevation	Time to return to normal
CPK MB	3-12hrs	24hrs	48-72hrs
CTnI	3-12hrs	24hrs	5-10 day
cTnT	3-12hrs	12hrs -2days	5-14 days

VIII. MANAGEMENT

1. Acetyl salicylic acid 325 mg to chewed once MI is suspected.
2. Supplemental oxygen 2 – 4 liters/min for 6-12 hours.
3. Control of chest pain:
    a) Analgesics: morphine repeated every 5 min in small doses (2-4mg)
    b) Nitrates can be given either sublingually or intravenously
    c) Beta adrenoreceptor blocker – metoprolol 5mg over 2-5 minutes for a total of three doses.
4. Thrombolysis – 1.5 million units of streptokinase to be infused intravenously over 1 hour.

5. ACE Inhibitor – important for both short and long term survival. The mortality benefits of ACE inhibitor is additive to aspirin and beta blockers.

6. Calcium antagonists – Nifedipine has no role. Verapamil and diltiazem can be given for relief of ongoing ischemia in patients for whom beta blockers are ineffective or contraindicated.

IX. DISTURBANCES ON CONDUCTION SYSTEM OF HEART IN MI

A. Arrhythmias

The mechanism of arrhythmias in acute phase of coronary occlusion is reentry due to inhomogenisity of the ischaemic myocardium (28). The other causes are electrolyte imbalance and autonomic nervous system imbalance. The reperfusion arrhythmia is due to wash out of various ions such as lactate, potassium and toxic substances that have accumulated in the ischaemic zone.

1. Ventricular Premature Beats

Infrequent sporadic ventricular premature beats occurs in almost all patients with MI and do not require therapy. QRS complex is bizarre, widened, slurred or notched. Compensatory pause is complete.

2. Ventricular Tachycardia and Fibrillation

Ventricular rhythm abnormalities are common during the early phase of acute MI with an incidence of ventricular fibrillation within first four hours, so called primary ventricular fibrillation, of 3-5% which declines rapidly thereafter. (29). Primary ventricular fibrillation is thought to be the result of micro entry mechanism in the infarct zone (30). Ventricular fibrillation is a major cause of death in those who die before receiving medical attention (31). Sustained VT which is hemodynamically stable should be treated with Intravenous lignocaine (bolus 1- 1.5mg/kg body weight along with maintenance infusion of 20-50mg/kg). If it does not stop promptly electrical cardio version should be attempted. For VF, unsynchronized DC shock of 200 – 300W/sec is used.

3. Sinus Bradycardia

It is relatively common early in the course of acute MI especially in inferior wall infarction and after reperfusion of right coronary artery because of the activation of vagal efferent that ultimately results in enhanced parasympathetic tone (32). Treatment is indicated if there is hemodynamic compromise. Atropine enhances the discharge rate of the sinus node and facilitates AV conduction (33). It also reverses the peripheral effects of excessive cholinergic activity.

4. Accelerated Idioventricular Rhythm

It frequently occurs during the first hour of acute MI and after thrombolysis as a reperfusion arrhythmia (34). It does not warrant treatment. However if rhythm speeds up to a rate of about 120 beats/min, it should be suppressed with lignocaine (35).

5. Atrial flutter/ fibrillation

Atrial flutter is uncommon in acute myocardial infarction, while atrial fibrillation has an incidence of 10 -15% (36). Atrial fibrillation is associated with increased in-hospital mortality rate as it is associated with large infarct and it seems relatively more common in older patients, those with cardiac failure, advanced AV block, atrial infarction. Rate can be controlled in atrial fibrillation by beta blockers verapamil and Digoxin. DC shock is employed if there is a hemodynamic compromise.

B. ATRIO – VENTRIVULAR CONDUCTION BLOCK

1. First Degree AV Block

ECG finding - prolongation of PR interval more than 0.2 seconds.

Common with acute myocardial infarction especially IWMI, due to Ischaemia or enhanced vagal activity. No therapy is necessary.

2. Second Degree AV Block

Intermittent failure of atrial impulses to conduct to the ventricle and exist in two forms.

a. Mobitz type I –

- Occurs as a result of ischemia of AV node.
- Most commonly seen in IWMI
- Associated with narrow QRS complex.
- Usually has varying PR interval with non conducted beat following a progressive lengthening of PR interval.

b. Mobitz type II –

- Lesion is in the conduction system below bundle of HIS.
- Associated with widening of QRS complex.
- Seen in AWMI
- Characterized by non varying PR interval before the non conducted atrial impulse.
- Potentially can progress to complete heart block.

3. Third Degree AV block

It is characterized by complete interruption of AV conduction. The atrium is thus activated by one pacemaker, usually sinus pacemaker

and ventricles by another. QRS complexes are narrow because the site of block being above His Bundle in about 70% cases. Complete heart block in IWMI is not an independent predictor of poor outcome as it is due to the ischemia of the AV node (34) and is usually transient. Patients with AW infarction who develops third degree block has a mortality of 80% as it reflects extensive infarction and extensive destruction of the conduction system (35).

4. Intraventricular Conduction Block

The presence of bundle branch block is associated with two fold increase in the hospital mortality rate compared with absence of bundle branch block (36).

The presence of bundle branch block in MI identifies patients who (37).

1. are more likely to develop CCF
2. More likely to develop high grade heart block
3. More likely to have an episode of VF
4. More likely to have higher mortality rate.

a) Left Anterior Fascicular Block

Features in ECG are (38)

- Abnormal left axis deviation (-45 to -60)
- ‘rs’ complex in leads II, III, aVF and ‘qR’ in leads I and aVL
- Delayed intrisicoid deflection in leads I & aVL
- Peak of R wave in Lead III occurring earlier than peak of R wave in lead II.
- Peak of R wave in Lead aVL occurring earlier than peak of R wave in lead aVR.

b) Left Posterior Fascicular Block

Features in ECG are

- Mean QRS axis more than 120°.
- RS pattern in leads I and aVL with qR pattern in inferior leads.
- Duration of QRS complex less than 120ms.
- Exclusion of other factors causing right axis deviation.

c) Right Bundle Branch Block

- QRS duration more than 120ms.
- Progressive diminution of S wave in V2 is the earliest sign (39).

- Progressive enlargement of R′or r′ with final widening of this deflection in V2.
- Wide and deep S wave in V5 and V6.
- The Q wave of infarct is not usually impeded by presence of RBBB.

d) Left Bundle Branch Block

1. QRS duration more than 120ms.
2. Broad notched R wave in V5, V6 and usually in lead LI , aVL.
3. Small or absent initial R wave in right precordial leads (V1, V2), followed by deep S wave.

Diagnosis of MI in presence of LBBB.

1. Sgarbossa suggested that ST segment elevation of 1 mm or more concordant with QRS polarity has a high sensitivity and specificity.
2. Appearance of ST elevation in lateral leads or ST depression with deep T wave inversion in V1 to V3.
3. presence of QR complexes in leads I, V5 ,V6 or leads II, III, aVF

Diagnosis of old MI in presence of LBBB

1. small Q waves in leads I, V5 ,V6 (40)
2. sign of Cabera and Friedland – a late notching of S wave in V3 through V5 (41)
3. Chapman's sign – notching of upstroke of R wave in leads I, aVL, V5, V6.

e) Bifascicular Block

The combination of RBBB with either left anterior or left posterior block is known as bifascicular block. The risk of developing complete AV block is quite high. Mortality is high because of occurrence of sever pump failure secondary to extensive myocardial necrosis (42)

f) Trifascicular Block

Trifascicular block implies involvement of all the three fascicles. It requires an ECG pattern of bifascicular block and evidence of prolonged AV conduction. 40% of patients with Trifascicular block progresses to complete AV block.

MATERIALS AND METHODS

The study was conducted in Government Royapettah Hospital during the period from January 2006 to may 2006. The patients admitted to ICCU were taken up for study.

A detailed history physical examination &laboratory work up was done in all patients.

Total No of Patients: 100

Age Group: 20 to 90

Clinical presentation

Enzyme levels

ECG findings

Echocardiography findings

were taken into account.

CRITERIA FOR ENTRY TO STUDY POPULATION

1. History of chest pain lasting more than 30 minutes not relieved by nitrates or rest.

2. Typical ECG changes of acute myocardial infarction

EXCLUSION CRITERIA

All patients with evidence of preexisting rhythm disturbances were excluded from the study.

VARIABLE RECORDED AT ADMISSION

ECG were taken in all patients

ECG recording were done immediately after admission, if thrombolysed one hour later, once daily for 7 days, whenever complications occurred & at discharge.

Special right precordial leads V3R V4R V5R were taken in patients with inferior wall MI & also posterior leads were taken in suspected cases of posterior wall MI

Long strips in L II, V I were taken to study the rhythm disturbances.

OBSERVATION

100 Patients of acute myocardial infarction were included in this study. They consisted of 77 male and 23 female patients. The youngest patient was a 23 year old male and the oldest patient was a 84 year old male.

1. INCIDENCE OF RHYTHM DISTURBANCES IN PATIENTS WITH ACUTE MI

No of patients with rhythm disturbances	No of patients without rhythm disturbances	Total patients
46	54	100

Out of 100 patients of AMI 46 patients had Rhythm disturbances.

2. INCIDENCE OF RHYTHM DISTURBANCES IN PATIENTS WITH ACUTE MI ACCORDING TO AGE

Age	**3rd decade**	**4th decade**	**5th decade**	**6th decade**	**7th decade**	**8th decade**	**9th decade**	**Total**
	20-29 Years	**30-39 years**	**40-49 years**	**50-59 years**	**60-69 years**	**70-79 years**	**80-89 years**	
Number of patients	2	3	20	32	28	11	4	100
Number of rhythm disturbances	2	1	6	17	13	6	1	46

The maximum incidence of MI is in the age group 50 to 59 years. The rhythm disturbances were also maximum in this age group. The minimum incidence of myocardial infarction was in the age group 20 to 29 years. The oldest patient was 85 year old male. The youngest patient was 23 year old male.

3. INCIDENCE OF RHYTHM DISTURBANCES IN PATIENTS WITH ACUTE MI ACCORDING TO SEX

Sex	Total (100)	Rhythm Disturbance	Percentage	p value
Male	80	37	46.25	0.3665
Female	20	9	45.0	

Out of 100 patients with myocardial infarction 80 were males & 20 were females. Out of 80 males 37 had rhythm disturbances with a percentage of 46.25 & out of 20 females 9 had rhythm disturbance with a percentage of 45.

4. INCIDENCE OF RHYTHM DISTURBANCES IN PATIENTS WITH VARIOUS RISK FACTORS IN ACUTE MI

Risk Factors	Patient with MI (Total =100)	Patient with Rhythm Disturbances		p value
	No. of Patients	No. of Patients	percentage	
Smoking	57	38	66.7	0.0000
Diabetes	55	37	67.3	0.0000
Hypertension	38	23	60.5	0.0225
Alcohol	33	25	75.8	0.0000
Hypercholesterolemia	51	19	37.3	0.0734

8 patients had previous ischemic heart disease prior to present episode. Of these 4 developed rhythm disturbances. All female patients were postmenopausal. 6 patients had COPD and 3 developed rhythm disturbances.

5. SITE OF INFARCTION

Site of Infarction	Number	Percentage
AWMI		61%
I. ASMI	43	
II. EAWMI	13	
III. ALMI	5	
IWMI	39	39%

The incidence of Anterior wall MI is more than inferior wall MI. The incidence of anterior wall myocardial infarction was 61% and that of inferior wall myocardial infarction was 39%.

6. INCIDENCE OF DIFFERENT TYPES OF RHYTHM DISTURBANCES IN ACUTE MYOCARDIAL INFARCTION

1) AV BLOCK

Type of AV Block	**1ST degree**	**2nd degree**	**3^{d} degree**	**Total**
No of patients	6	6	6	18

All second degree blocks were Mobitz type 1 & there were no patients with Mobitz type two block. Out of 100 patients there were 18 patients with AV block.

2) INTRAVENTRICULAR BLOCK

Type of Intraventricular Block	No of Patients
LBBB	7
RBBB	10
LAFB	6
LPFB	0
BB	2
TOTAL	25

Out of 100 patients 25 patients had Intraventricular block, of these right bundle branch block was common with an incidence of 10.

7. INCIDENCE OF OTHER RHYTHM DISTURBANCES. IN ACUTE MYOCARDIAL INFARCTION

The other arrhythmias associated with acute myocardial infarction were ventricular ectopic beat, atrial fibrillation, ventricular tachycardia, ventricular fibrillation whose incidence are as follows:

Type of Arrhythmias	No of Patients
Ventricular ectopic beat	24
Atrial fibrillation	7
Ventricular fibrillation	2
Ventricular tachycardia	8

Sinus tachycardia was noted in 43 patients immediately after admission. It could be attributed to stress and anxiety leading to increased sympathetic activity. Hence it was not included as rhythm disturbances in our study.

8. INCIDENCE OF RHYTHM DISTURBANCES IN RELATION TO LOCATION OF MYOCARDIAL INFARCTION

Location of MI	No. of patients (total = 100)	No. of patients with rhythm disturbances	Percentage	P value
AWMI	61	39	50.8	0.2265
IWMI	39	15	38.5	

Out of 61 patients with anterior wall MI 39 patients had rhythm disturbances with a percentage of 50.8. Out of 39 patients with inferior wall MI 15 had rhythm disturbances with a percentage of 38.5.

2) INCIDENCE OF AV CONDUCTION BLOCK IN RELATION TO LOCATION OF MYOCARDIAL INFARCTION

Location of MI	1st degree	2nd degree	3rd degree	Total AV Block	P value
IWMI	2	5	4	11	0.0507
AWMI	4	1	2	7	
Total	6	6	6	18	

The number of patients with AV block in inferior wall MI was 11 while in anterior wall MI was 7. AV block is more common in IWMI than AWMI

3) INCIDENCE OF INTRAVENTRICULAR CONDUCTION BLOCK IN RELATION TO LOCATION OF MYOCARDIAL INFARCTION

Location of MI	RBBB	LBBB	LAFB	BB	Total
AWMI	8	5	5	2	20
IWMI	2	2	1	0	5
TOTAL	10	7	6	2	25

Out of 100 patients with MI, There were 25 patients with Intraventricular conduction block .The total number of patients with Intraventricular conduction block in anterior wall MI was 20 & in

inferior wall MI was 5.Thus Intraventricular conduction block is more common in anterior wall MI .

3) INCIDENCE OF OTHER RHYTHM DISTURBANCES IN RELATION TO LOCATION OF MYOCARDIAL INFARCTION

Location of MI	Ventricular Ectopic	Ventricular tachycardia	Atrial Fibrillation	Ventricular Fibrillation	Total
AWMI	17	8	6	2	33
IWMI	7	0	1	0	8
TOTAL	24	8	7	2	41

The incidence of other arrhythmias associated with acute myocardial infarction such as ventricular ectopic beat , atrial fibrillation ventricular tachycardia, ventricular fibrillation were also high in anterior wall MI .

9. TIME OF APPEARANCE OF RHYTHM DISTURBANCES IN RELATION TO ONSET OF ACUTE MI

Type of Block	Time of Appearance			
	At admission	Within 1st 24 hours	2-5 days	>5 days
1st degree AV block	3	2	0	1
2nd degree AV block	3	2	1	0
3rd degree AV block	4	2	0	0
RBBB	5	2	2	1
LBBB	3	3	1	0
LAFB	3	1	1	0
OTHERS	1	2	1	0

Most of the rhythm disturbances occurred within first 24 hours.

10. INCIDENCE OF RHYTHM DISTURBANCES IN RELATION TO THROMBOLYSIS

	No of patients	No of patients with rhythm disturbances	Percentage	P value
Thrombolysed	52	17	32.7	0.0054
Non thrombolysed	48	29	60.4	

Out of 100 patients with MI 52 were thrombolysed. Among 48 non thrombolysed patients 29 developed rhythm disturbances with a percentage of 60.4.Among thrombolysed patients 17 developed rhythm disturbances with a percentage of 32.7.

11. OUTCOME

Outcome	No of patients (total = 100)	No of patients with rhythm disturbances	Percentage	P value
Improved	79	31	39.2	0.0085
Died	21	15	71.4	

The total mortality was 21 %. Among 21 died 15 patients had rhythm disturbances.

11. MORTALITY AMONG ANTERIOR & INFERIOR WALL MI

Location of MI	No of Patients	No of Death	Percentage
AWMI	61	18	29.50
IWMI	39	3	7.69

The mortality rate among 61 patients with anterior wall MI is 29.50. The mortality rate among 39 patients with inferior wall MI IS 7.69.

12. DISTRIBUTION OF DEATH ACCORDING TO TYPE OF RHYTHM DISTURBANCES

AV BLOCK	No. of patients	No of death	Percentage
1st degree	6	0	0
2nd degree	6	0	0
3rd degree	6	3	50

The mortality rate among 3rd degree AV block patients was 50%.

Intraventricular Block	No. of Patients	Death	Percentage
LAFB	6	2	33.33
RBBB	10	3	30
LBBB	7	3	42.85
BB	2	1	50
VT	8	5	62.5
VF	2	2	100

There were no deaths among 1& 2 AV degree block. Out of 100 MI patients 6 developed 3rd degree AV block; among them 3 died. The percentage of mortality among patients developing LBBB, LAFB, VT, and BB were 42.85, 33.33, 62.5, 50 respectively.

DISCUSSION

1. AGE AND SEX

In this study that included 100 patients of acute myocardial infarction, the maximum incidence of acute myocardial infarction occurred in the age group 50 to 59. The rhythm disturbances were also maximum in this age group. The youngest patient was a 23 year old male. The oldest patient was an 84 year old male. With increasing age there was a greater incidence of death in those with arrhythmia; despite the fact that the incidence of serious arrhythmias remains relatively constant at various ages. (43)

In this study male patient constituted 80% 0f study group. Female population constituted 20% of study group. According to literature males in the age group 40 to 70 are prone for myocardial infarction (44) which correlates with our study.

Among 80 males with MI 37 developed rhythm disturbances with a percentage of 46.25.Among 20 females 9 had developed rhythm disturbances with a percentage of 45. p value was 0.3665 ,hence they were not statistically significant. The proportion of patients with rhythm disturbances appeared to be almost equal in both sexes. All females with rhythm disturbances were post menopausal.

2. INCIDENCE OF RHYTHM DISTURBANCAS IN PATIENTS WITH RISK FACTORS

Smoking was present in 57 patients among 100 with Acute MI. It is the most common risk factor in our study group. Diabetes is the second most common risk factor with an incidence of 55%. HARRIS et al found that coronary heart disease accounts for 69%of death in adults with diabetes.

In this study group the total incidence of hypertension was 38%. When serum cholesterol above 200mg% is taken as hypercholesterolemia, the incidence of this risk factor was 51%.According to LAW et al a 10% increase in cholesterol is associated with 20 to 30% increase in risk of coronary artery disease.

38 patients with smoking developed rhythm disturbances with a percentage of 66.6.Among diabetic patients 37 developed rhythm disturbances with a percentage of 67.3 .The correlation between smoking ,diabetes mellitus & rhythm disturbances was found to be statistically significant with a P value of 0.000.

The correlation between alcohol consumption & rhythm disturbances was also found to be statistically significant with a P value of 0.000 as the incidence of alcohol consumption among 100 patients

with MI was 38 & the percentage developing rhythm disturbances among them was 75.8.

19 patients with hypercholesterolemia developed rhythm disturbances with a percentage of 37.3. In our study the association between hypercholesterolemia and rhythm disturbances was not statistically significant as the P value was 0.0734.

Study shows that multiple risk factors are associated with increase in the risk of rhythm disturbances. 58% of our patients with rhythm disturbances had multiple risk factors.8 patients had previous history of coronary heart disease and 4 of them developed rhythm disturbances .6 patients had chronic obstructive pulmonary disease and 3 developed rhythm disturbances.

3. INCIDENCE OF RHYTHM DISTURBANCAS IN RELATION TO LOCATION OF ACUTE MYOCARDIAL INFARCTION

In this study of 100 patients with acute MI 46 developed some form of rhythm disturbances .Among 61 patients with anterior wall MI 39 developed rhythm disturbances with a percentage of 50.8 and in 39 patients with inferior wall MI 15 developed rhythm disturbances with a percentage of 38.5. In our study the relation between anterior wall MI and inferior wall MI and the rhythm disturbances was not statistically

significant as the P value was 0.2265.Thus in our study rhythm disturbances occurred equally in both anterior wall and inferior wall MI

According to the literature the overall incidence of atrioventricular conduction blocks in acute MI is 12 to 25 %.The incidence of 1st degree AV block is 4 to 14 % , 2nd degree AV block is 6 to 10 %(45), and 3rd degree AV block is 5 to 8% (46). AV blocks are more common in inferior wall MI than in anterior wall MI (46). If it occurs in anterior wall MI it is associated with extensive myocardial necrosis and signifies poor prognosis.

The results of our study coincides well with the literature. In our study the incidence of atrioventricular conduction block was 18% .The incidence of 1st degree, 2nd degree, 3rd degree AV blocks were 6, 6, and 6 respectively. There were 11 patients with inferior wall MI developing AV block and 7 patients with anterior wall MI developing AV blocks (p value 0.05). Thus from our study it was concluded that AV blocks are more common in inferior wall MI than in anterior wall MI.

Most of the 1st & 2nd degree AV block developed on the first day of the MI and all of them disappeared before discharge.

According to BRAUNWALD'S TEXT BOOK OF CARDIOLOGY the incidence of MOBITZ type two block is less than 1% .In our study there was no patient with MOBITZ type two block The

high incidence of AV block in inferior wall MI is related to the blood supply of the AV node from the branch of the dominant coronary artery, specifically the right coronary artery in 90%of patients.

In our study 25% of patients developed some form of Intraventricular conduction block, which correlates well with literature which says Intraventricular conduction block occurs in about 10 to 20 % (47) In the study of Atkins &his group the most common abnormal conduction pattern was RBBB followed by LAFB LBBB . In our study the incidence of RBBB, LAFB LBBB were 10, 6, and 7 respectively. Thus RBBB is the most common abnormal conduction pattern.

Two patient developed bifascicular block .No patient developed left posterior fascicular block

Thus the anatomical characteristic of right bundle branch and anterior fascicle of left bundle branch their length and slenderness make them more vulnerable to ischaemic injury than the more compact posterior fascicle of left bundle branch. Their initial common course and blood supply account for the high incidence of RBBB and LAFB in the setting of acute MI. Most of the Intraventricular conduction block occurred in patients with anterior wall MI (no of patients 20). In inferior wall MI the no of patients with Intraventricular conduction block is 5.

Other arrhythmias which occurred in our study were ventricular ectopic, atrial fibrillation, ventricular tachycardia and ventricular fibrillation. According to Ledda A et al the incidence of atrial fibrillation is 7 to 18 % in patients with acute MI (48). In our study the incidence of atrial fibrillation is 7 %(6 in AWMI, 1 in IWMI)

In our study group ventricular ectopic was the most common rhythm disturbances with an incidence of 24%. Ventricular tachycardia is seen in 8% of cases ,all of them occurring in anterior wall MI .The incidence of Ventricular fibrillation in patients with acute MI is 5%(49). In our study it is 2% which occurred in patients with extensive anterior wall MI

4. MORTALITY RATE IN ACUTE MI IN RELATION TO RHYTHM DISTURBANCAS.

The total mortality rate in our study population was 21%.According to Millon et al the incidence of death in acute MI is 24%.Among 79 patients who improved 31 had rhythm disturbances making a percentage of 39.2. Among 21 patients who died 15 had rhythm disturbances making a percentage of 71.4.Therefore the study clearly shows that rhythm disturbances is associated with significant mortality (p value 0.0085).

The incidence of 1st, 2nd, 3rd degree AV block are 6, 6, and 6, respectively. No deaths occurred in patients with 1st and 2nd degree AV block. This shows that development of 1st and 2nd degree AV block does not adversely affect prognosis. Six patients had 3rd degree AV block , among them 3 died making a percentage of 50%.Two patients with extensive anterior wall MI developed 3rd degree block ,both of them died .Strasberg and his associates found that patients with advanced AV block had lower left and right ventricular ejection fraction and higher left and right ventricular wall motion abnormality on two dimensional echocardiography than those who do not.

The mortality rate among IWMI is 7.69 and among AWMI is 29.50. Patients with IWMI who developed 3rd degree block have an increased mortality rate when compared to those who do not. Improved survival with pacing probably occurs in only small proportion of patients with complete AV block and anterior wall MI because of the extensive myocardial necrosis that almost invariably accompany this condition.

IN our study the mortality associated with development of LBBB, LAFB, RBBB, BB ARE 42.85, 33.33, 30, and 50 respectively. According to ROOS & DUNNING, HIDMAND et al the incidence of death in LBBB, RBBB, and LAFB is 44.7,29,48 respectively.

Ventricular tachycardia is associated with highest incidence of death of61 %(49).In our study there were 8 cases of ventricular tachycardia. All of them occurred in patients with anterior wall MI .Out of 8 patient 5 died making a percentage of 62.5.Two of the patients with extensive anterior wall MI developed ventricular fibrillation . Both of them died within first 24 hours.

5. THROMBOLYSIS AND RHYTHM DISTURBANCES

Out of 100 patients 52 were thrombolysed and 48 were not. Among thrombolysed 17 had rhythm disturbances and among non thrombolysed 29 had rhythm disturbances making a percentage of 60.4. Thus thrombolysis is associated with lesser incidence of rhythm disturbances.

CONCLUSION

In this dissertation entitled the "**THE STUDY OF RHYTHM DISTURBANCES IN ACUTE MYOCARDIAL INFARCTION IN GOVERNMENT ROYAPETTAH HOSPITAL**" 100 patients with acute myocardial infarction were studied with reference to clinical presentation, risk factors, ECG findings, and echocardiographic finding and observed rhythm disturbances were correlated and following conclusions were derived:

1) The incidence of rhythm disturbances in acute myocardial infarction is 46%.

2) The distribution of rhythm disturbances are almost equal in males and females (p value 0.3365)

3) Rhythm disturbances were more common in the age group 50-59

4) The incidence of rhythm disturbances in patients with Diabetes mellitus and hypertension is high. (p value 0.0000)

5) The incidence of rhythm disturbances in patients who smoke is high. (p value 0.0000)

6) The incidence of rhythm disturbances in patients who consume alcohol is high. (p value 0.0000)

7) Atrioventricular blocks are more common in inferior wall myocardial infarction. (p value 0.0507)

8) Ventricular ectopics, ventricular tachycardia, ventricular fibrillation, atrial fibrillation are more common in anterior wall myocardial infarction

9) Most of the Intraventricular conduction block occurred in patients with anterior wall myocardial infarction.

10) Right bundle branch block is the most common Intraventricular conduction abnormality noted with an incidence of 10.

11) The incidence of rhythm disturbances in non thrombolysed patients is more (29%) compared to thrombolysed patients 17% (p value 0.00545)

12) The incidence of death is increased among patients with rhythm disturbances (p value 0.00853).

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PROFORMA

NAME AGE SEX
OCCUPATION IPNO
DOA DOD

SYMPTOMS

ANGINA
SWEATING
PALPITATION
BREATHLESSNESS
VOMITING
LOSS OF CONSCIOUSNESS

PAST HISTORY

ISCHAEMIC HEART DISEASE
DIABETES MELLITUS
HYPERTENSION
CHRONIC OBSTRUCTIVE PULMONARY DISEASE

PERSONAL HISTORY

Smoking, alcohol

MENSTRUAL HISTORY

Premenstrual /postmenstrual

GENERAL EXAMINATION

CYANOSIS
ANAEMIA
SWEATING
PEDAL EDEMA

VITALS:

PULSE **:**RATE , RHYTHM , CHARACTER
BLOOD PRESSURE
RESPIRATORY RATE

TEMPERATURE
JUGULAR VENOUS PRESSURE

EXAMINATION OF CVS

APICAL IMPULSE: POSITION , THRILL
PALPABLE SOUNDS
S1- NORMAL/LOUD/SOFT/SPLIT
S2- NORMAL SPLIT/ ABNORMAL SPLIT
MURMUR
RUB
GALLOP

RESPIRATORY SYSTEM

BREATH SOUNDS
ADDED SOUNDS

ABDOMEN

ORGANOMEGALY ; FREE FLUID

CENTRAL NERVOUS SYSTEM

FND

INVESTIGATIONS

UREA/SUGAR/CREATINE /ELECTROLYTE
CPK
SERUM CHOLESTEROL
ECG WITH RHYTHM STRIP
X-RAY CHEST PA VIEW
ECHO

TREATMENT

THROMBOLYSED OR NOT

RHYTHM DISTURBANCES

TYPE
TIME OF APPEARANCE

OUTCOME

KEY TO MASTER CHART

ALC	-	alcohol
ALMI	-	anterolateral myocardial infarction
ASMI	-	Anterosptal myocardial infarction
AV BLOCK	-	atrioventricular block
BR	-	breathlessness
CC NO	-	cardiac care number
COPD	-	chronic obstructive pulmonary disease
CP	-	chest pain
CPK MB	-	creatine phosphokinase MB isoenzyme
DM	-	diabetes mellitus
EAWMI	-	extensive anterior wall myocardial infarction
ECG	-	electrocardiogram
ECHO	-	echocardiogram
F	-	Female
HT	-	hypertension
IHD	-	ischemic heart disease
IVCB	-	Intraventricular conduction block
IWMI	-	inferior wall myocardial infarction
M	-	Male
Pa. His	-	part history

PAL	-	palpitation
Pres .comp	-	presenting complaints
RVMI	-	right ventricular myocardial infarction
SCHO	-	serum cholesterol
SM	-	smoking
SW	-	sweating
SYN	-	syncope
THRO	-	thrombolysis
VOM	-	vomiting

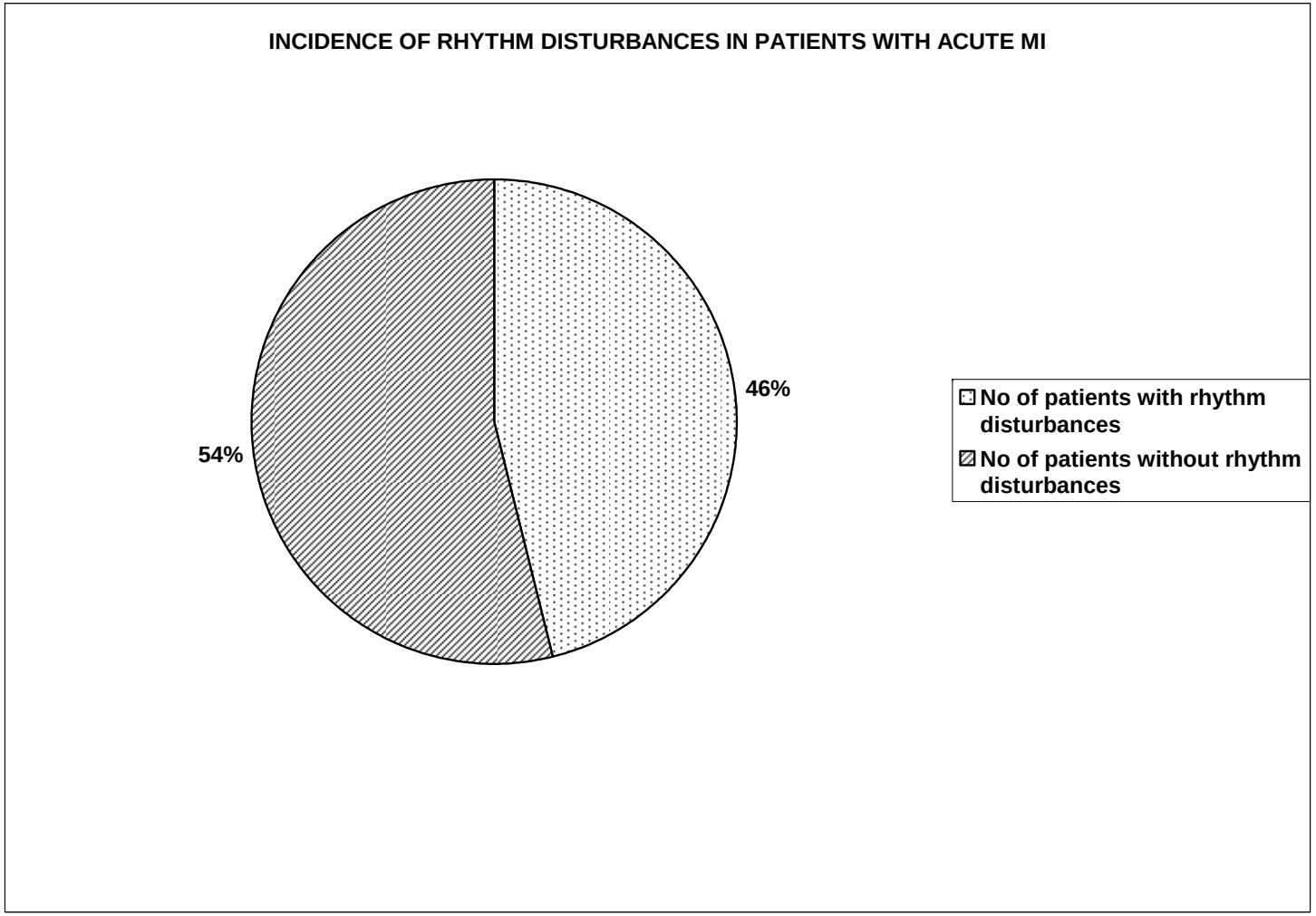
MASTER CHART

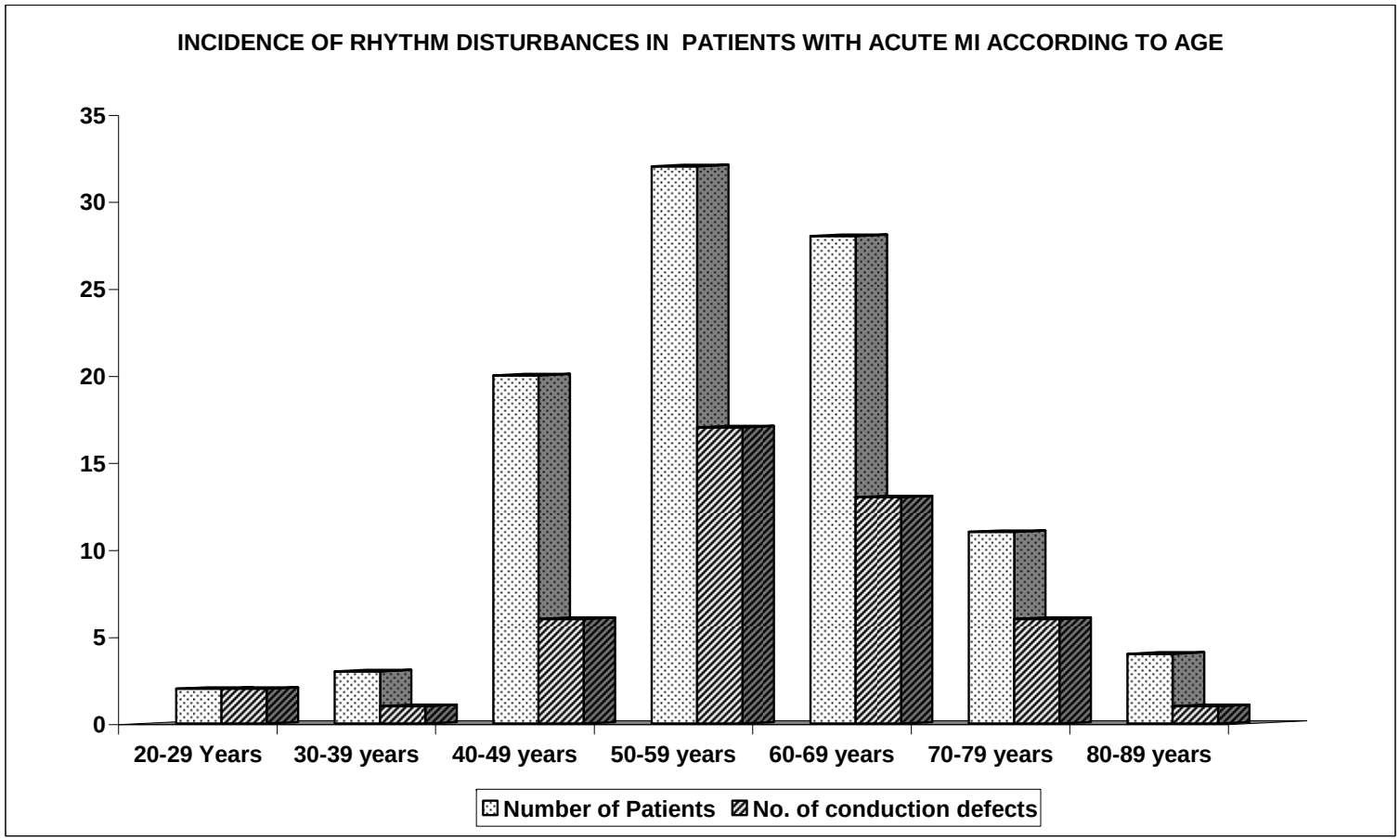
Sl NO.	Name	CC NO	Age	Sex	Pres. Comp	DM	HT	Pa.His	CPK	S CHO	ECG	ECHO LVAV	BLOCK	IV CB	OTHERS	RYH DIS	THRO	OUTCOME
1	PANDIAN	001	75	M	CP	NO	NO	NIL	292	195	IWMI	YES	NO	NO	NO	NO	YES	IMPROVED
2	VELU	003	62	M	CP	YES	YES	SM	291	220	IWMI RVMI PWMI	YES	2	NO	VPC	YES	NO	IMPROVED
3	LAKSHMI	006	52	F	PAL	YES	YES	NIL	364	184	ASMI	YES	3	LAFB	NO	YES	YES	IMPROVED
4	GRACES	007	61	F	CP	YES	NO	NIL	428	188	IWMI	YES	2	NO	VPC	YES	NO	IMPROVED
5	SEKAR	010	467	M	CP	NO	NO	SM	370	176	IWMI	NO	NO	NO	NO	NO	YES	IMPROVED
6	PADMA	026	60	F	CP	YES	NO	NIL	424	152	ASMI	YES	NO	NO	NO	NO	YES	IMPROVED
7	LOGAMMA	020	75	F	SYN	NO	YES	SM/ALC	436	168	ASMI	YES	NO	RBBB	AF	YES	NO	IMPROVED
8	RAJAMA	023	50	F	CP	NO	NO	NIL	512	153	IWMI	NO	NO	NO	NO	NO	YES	IMPROVED
9	MEENAKSHI	024	84	F	CP BR	NO	YES	NIL	344	194	IWMI PWMI	YES	NO	NO	NO	NO	NO	IMPROVED
10	SRINIVASAN	028	62	M	CP	YES	NO	NIL	428	248	EAWMI	YES	NO	NO	NO	NO	YES	IMPROVED
11	LATHA	029	63	F	CP	YES	YES	SM/ALC	414	215	IWMI	NO	2	NO	VPC	YES	NO	IMPROVED
12	SAM	040	44	M	CP SYN	NO	YES	NIL	290	208	IWMI RVMI	YES	NO	NO	NO	NO	YES	IMPROVED
13	MANI	044	40	M	CP SW	NO	YES	SM	312	194	IWMI RVMI PWMI	YES	NO	NO	NO	NO	NO	IMPROVED
14	ISMAIL	048	60	M	CP	YES	NO	SM	256	238	ASMI	YES	NO	NO	NO	NO	NO	IMPROVED
15	SAMBASIVAM	054	40	M	CP SW	NO	NO	IHD	283	242	EAWMI	YES	NO	NO	NO	NO	YES	IMPROVED
16	VEMBU	058	68	F	CP	NO	YES	SM/ COPD	315	210	ASMI	YES	NO	NO	AF	YES	NO	IMPROVED
17	RAJA	070	60	M	CP	YES	NO	IHD	365	208	EAWMI	YES	NO	LAFB	VPC/ST	YES	NO	IMPROVED
18	CHANDRA MOHAN	077	52	M	CP	YES	YES	SM	345	245	IWMI	YES	3	NO	NO	YES	YES	DIED
19	SUBHU	078	80	F	SW PAL	YES	YES	NIL	340	198	IWMI RVMI PWMI	YES	NO	NO	NO	NO	YES	IMPROVED
20	VENKATACHALAM	088	72	M	BR	NO	NO	SM	438	180	EAWMI	YES	NO	NO	NO	NO	NO	IMPROVED
21	SRINIVASAN	109	63	M	CP	YES	NO	SM/ALC	12	288	EAWMI	YES	NO	RBBB	VT	YES	NO	DIED
22	RAM	128	30	M	CP BR	YES	YES	SM	370	220	EAWMI	YES	3	NO	VT	YES	NO	DIED
23	BALAMURUGAN	139	53	M	CP	NO	NO	SM	350	240	IWMI	YES	NO	NO	NO	NO	YES	IMPROVED
24	RAMALINGAM	147	53	M	CP	NO	NO	COPD	616	180	IWMI	YES	NO	NO	NO	NO	YES	IMPROVED
25	DURAI	154	48	M	CP PAL	NO	YES	NIL	672	210	IWMI PWMI	YES	NO	NO	NO	NO	YES	IMPROVED
26	KUPPUSWAMY	159	48	M	BR	YES	YES	SM	615	290	IWMI	YES	1	NO	AF	YES	YES	IMPROVED
27	RAMANATHAN	167	50	M	SW BR	NO	NO	SM	510	160	IWMI	YES	NO	NO	NO	NO	YES	IMPROVED
28	DHANA	178	65	F	CP	YES	NO	IHD	541	240	IWMI	YES	NO	NO	NO	NO	YES	IMPROVED

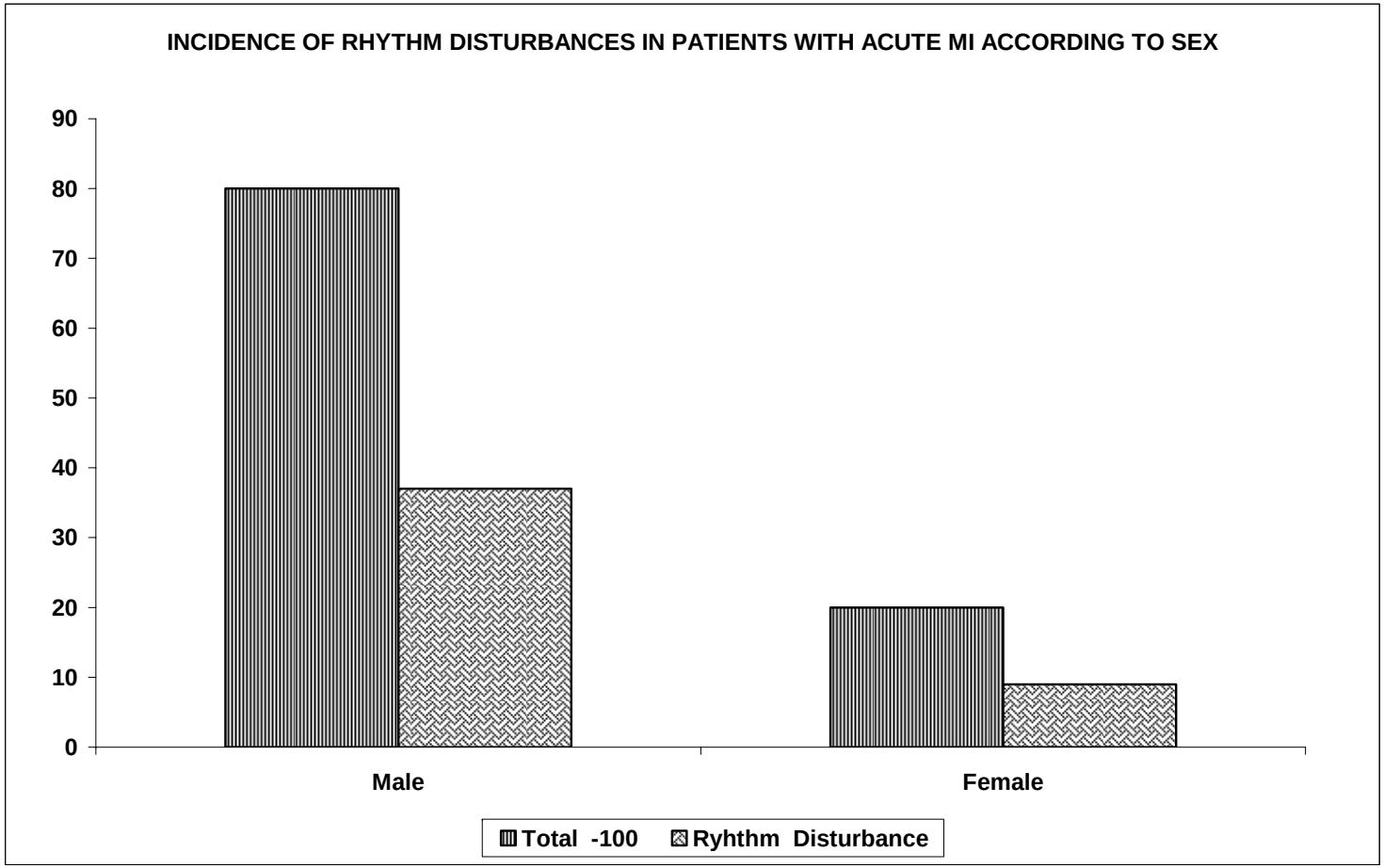
SI NO.	Name	CC NO	Age	Sex	Pres. Comp	DM	HT	Pa.His	CPK	S CHO	ECG	ECHO LVAV	BLOCK	IV CB	OTHERS	RYH DIS	THRO	OUTCOME
29	SULAIMAN	182	67	M	CP	NO	YES	ALC	430	230	ASMI	YES	1	BB	VT	YES	NO	DIED
30	VANITHA	185	58	F	CP	YES	NO	SM/ALC	312	260	IWMI	NO	NO	NO	AF	YES	YES	IMPROVED
31	VENUGOPAL	199	45	M	CP BR	YES	NO	NIL	398	185	IWMI	YES	NO	NO	NO	NO	YES	IMPROVED
32	SALAMON	208	50	M	CP	NO	YES	SM/ALC	290	195	IWMI	YES	NO	NO	NO	NO	YES	IMPROVED
33	BARGHAT NISHA	209	54	M	CP	NO	NO	NIL	318	185	ASMI	YES	NO	NO	NO	NO	YES	DIED
34	SHANMUGHAM	210	53	M	CP BR	YES	YES	SM ALC	412	208	ASMI	YES	NO	NO	VF	YES	NO	DIED
35	TAMILMANI	221	59	M	CP SW	NO	NO	SM /ALC	470	210	ASMI	YES	NO	RBBB	VPC	YES	NO	IMPROVED
36	SUBRAMANI	225	52	M	CP	NO	NO	SM COPD	558	190	ASMI	YES	NO	RBBB	NO	YES	NO	DIED
37	RAMYA	226	50	F	CP	YES	YES	SM/ALC	612	165	EAWMI	YES	NO	NO	VT	YES	NO	DIED
38	NARAYANAN	227	42	M	CP	YES	NO	SM/ALC	220	158	ASMI	YES	NO	RBBB	AF	YES	YES	IMPROVED
39	NAGARAJ	228	68	M	CP SW	YES	NO	SM	508	320	IWMI	YES	NO	NO	NO	NO	YES	IMPROVED
40	RAJINI	229	55	M	CP BR	NO	NO	SM/ALC	412	188	ASMI	YES	NO	NO	NO	NO	YES	DIED
41	VALARMATHY	232	60	F	CP	NO	YES	NIL	380	175	ASMI	NO	NO	NO	NO	NO	NO	DIED
42	THANGARAJ	234	59	M	CP	YES	NO	SM/ALC	450	165	IWMI	YES	1	NO	VPC	YES	NO	IMPROVED
43	DHARANI	254	75	M	CP SW	YES	NO	SM IHD	360	206	ASMI	YES	1	RBBB	AF	YES	NO	IMPROVED
44	KALAM	255	60	M	CP BR	NO	NO	ALC	528	258	ASMI	YES	1	BB	VPC	YES	NO	IMPROVED
45	VALAVANDHA	256	65	F	CP BR	NO	YES	NIL	612	165	IWMI	NO	NO	NO	NO	NO	NO	IMPROVED
46	KANAGAM	257	65	F	CP	YES	NO	SM/ALC	295	180	IWMI PWMI	YES	NO	NO	AF/VPC	YES	YES	IMPROVED
47	KUMARASEN	258	65	M	CP SW	NO	YES	NIL	353	236	ASMI	YES	NO	NO	NO	NO	NO	IMPROVED
48	DHANAPAL	259	70	M	CP	YES	NO	SM	618	175	ASMI	YES	NO	LAFB	VPC	YES	YES	IMPROVED
49	NAGARAJ	261	54	M	CP	NO	NO	SM/ALC	308	173	EAWMI	YES	NO	NO	NO	NO	NO	IMPROVED
50	PILLAI	264	67	M	CP	YES	YES	IHD/SK	510	160	IWMI PWMI	YES	3	NO	VPC	YES	YES	IMPROVED
51	VADIVELAN	269	30	M	CP SW	NO	NO	NIL	670	274	IWMI RVMI	YES	NO	NO	NO	NO	NO	IMPROVED
52	KUMARAVEL	270	60	M	CP	NO	NO	SM COPD	360	172	ASMI	YES	NO	NO	NO	NO	YES	IMPROVED
53	DEEPA	271	55	F	CP	YES	YES	SM	412	212	IWMI PWMI	YES	NO	RBBB	VPC	YES	NO	DIED
54	GANESAN	275	26	M	CP	YES	NO	IHD	554	216	ASMI	YES	NO	NO	VT	YES	YES	IMPROVED
55	GOPAL	278	72	M	CP SW	NO	NO	SM/ALC	250	234	EAWMI	YES	NO	NO	NO	NO	NO	DIED
56	MUNI	281	50	M	CP	NO	YES	NIL	294	164	ALMI	YES	NO	NO	NO	NO	YES	IMPROVED
57	MANI	283	65	M	CP	NO	YES	SM	368	178	ASMI	YES	NO	NO	NO	NO	NO	IMPROVED
58	MAGESH	286	48	F	CP	NO	NO	NIL	390	164	ASMI	YES	NO	NO	NO	NO	YES	IMPROVED

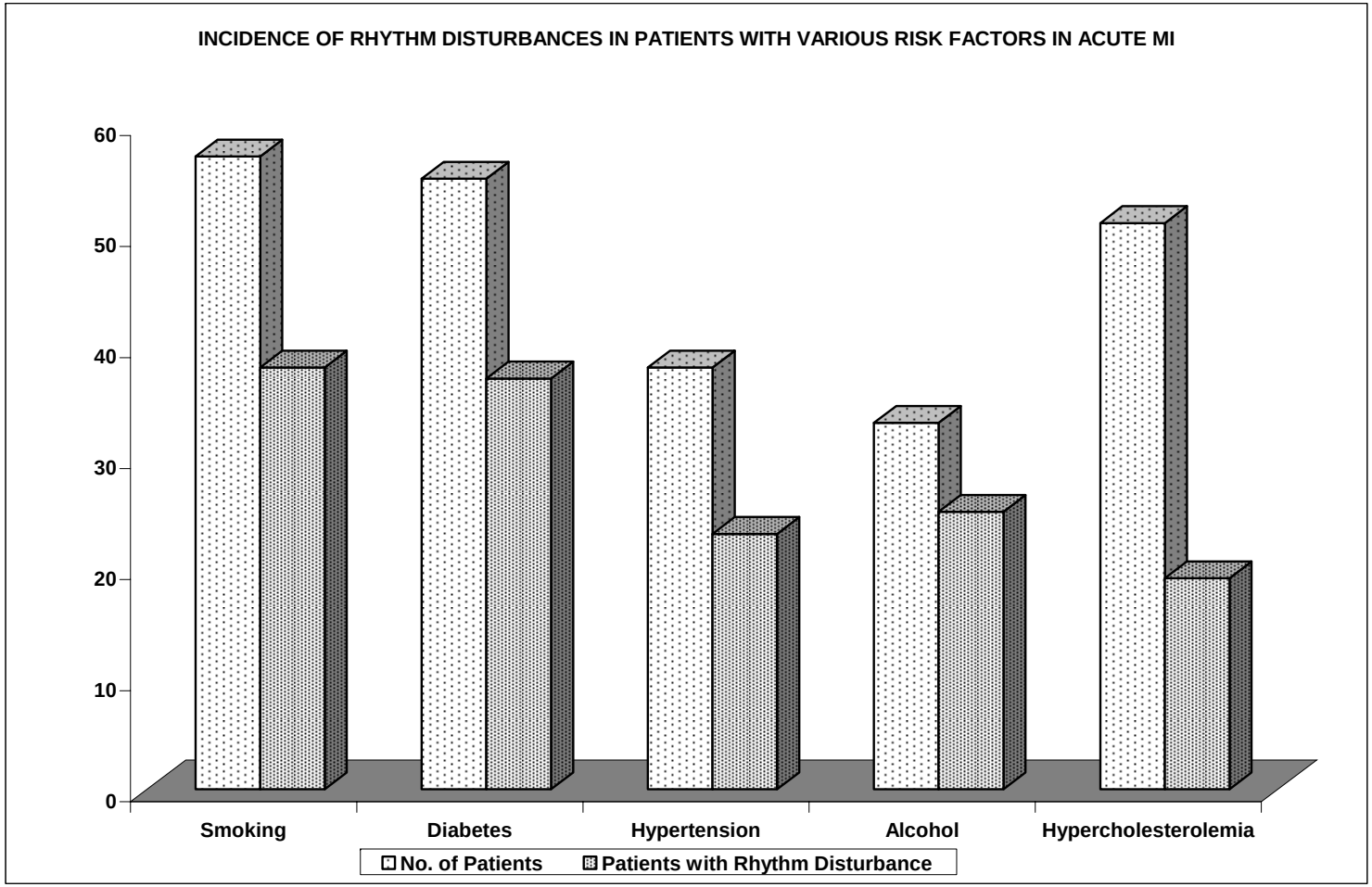
SI NO.	Name	CC NO	Age	Sex	Pres. Comp	DM	HT	Pa.His	CPK	S CHO	ECG	ECHO LVAV	BLOCK	IV CB	OTHERS	RYH DIS	THRO	OUTCOME
59	THANIGACHALAM	288	42	M	CP SW	YES	NO	SM/ALC	486	220	ALMI	YES	NO	LAFB	AF	YES	NO	IMPROVED
60	VETRIVEL	289	58	M	CP BR	YES	NO	SM/ALC	219	175	IWMI	YES	3	LBBB	VPC	YES	NO	IMPROVED
61	MARAN	290	50	M	BR	NO	NO	NIL	318	180	ASMI	YES	NO	NO	NO	NO	NO	DIED
62	SIVA	291	64	M	CP	NO	NO	SM ALC	424	162	ASMI	YES	NO	NO	NO	NO	NO	IMPROVED
63	ANEES AHMED	292	60	M	CP SW	YES	YES	SM/ALC	560	225	ASMI	YES	NO	LBBB	VPC	YES	YES	DIED
64	VARUN	293	56	M	CP	NO	NO	SM/ALC	500	284	EAWMI	YES	NO	NO	NO	NO	YES	IMPROVED
65	AHMED	298	85	M	CP	NO	NO	NIL	475	192	ASMI	YES	NO	NO	NO	NO	NO	IMPROVED
66	PERUMAL	300	55	M	CP BR	YES	NO	SM	380	210	IWMI	YES	3	LAFB	VPC	YES	YES	DIED
67	SELVADURAI	301	75	M	BR	YES	NO	SM COPD	345	158	IWMI	NO	NO	NO	NO	NO	YES	IMPROVED
68	RAJINI	302	23	M	SYN	YES	YES	SM/ALC	570	193	ASMI	YES	NO	LBBB	VPC	YES	YES	IMPROVED
69	BASKAR	303	43	M	CP SW	NO	NO	NIL	278	204	IWMI	NO	NO	NO	NO	NO	YES	IMPROVED
70	POONGAVANAM	305	50	M	CP	YES	YES	SM COPD	400	227	IWMI	YES	2	LBBB	NO	YES	YES	IMPROVED
71	RAMESH	307	47	M	CP	NO	YES	SM	345	234	ASMI	YES	NO	RBBB	VPC	YES	YES	IMPROVED
72	SIVA	308	45	M	CP	YES	NO	NIL	412	212	ASMI	YES	NO	NO	NO	NO	YES	IMPROVED
73	BAMA	310	65	F	CP	YES	YES	SM/ALC	340	178	EAWMI	YES	NO	NO	VF	YES	NO	DIED
74	KUPPUSWAMY	318	70	M	CP BR	YES	NO	SM/ALC	480	165	ASMI	YES	NO	LAFB	VPC	YES	NO	IMPROVED
75	ARASAN	319	67	M	CP	NO	NO	NIL	294	241	ASMI	YES	NO	NO	NO	NO	NO	IMPROVED
76	MANOHARAN	320	43	M	CP	YES	NO	SM/ALC	320	202	IWMI	YES	NO	NO	VPC/AF	YES	NO	IMPROVED
77	VINODHA	327	75	F	BR	NO	YES	IHD	360	198	IWMI	YES	NO	NO	NO	NO	YES	IMPROVED
78	ARAVINDH	328	55	M	SW	YES	NO	SM/COPD	500	225	ASMI	YES	NO	LBBB	VPC	YES	NO	IMPROVED
79	VIJAYKUMAR	329	55	M	CP VOM	YES	YES	SM	370	172	ASMI	YES	2	NO	VPC	YES	NO	IMPROVED
80	THIGARAJAN	330	50	M	CP SW	YES	NO	SM/ALC	421	184	ASMI	YES	NO	LBBB	VPC	YES	YES	IMPROVED
81	PONUSWAMY	331	75	M	CP	NO	YES	ALC	520	210	ASMI	YES	NO	NO	VT	YES	YES	DIED
82	RAMACHANDRAN	335	40	M	CP	YES	NO	SM/ALC	700	251	ASMI	YES	NO	NO	NO	NO	YES	IMPROVED
83	THANGAVELU	336	52	M	CP VOM	YES	NO	NIL	525	176	ASMI	YES	NO	NO	NO	NO	YES	IMPROVED
84	SEKAR	337	82	M	CP	NO	YES	SM	691	216	ASMI	YES	NO	RBBB	VPC	YES	NO	IMPROVED
85	RAVI	339	55	M	CP SW	NO	NO	NIL	578	174	ASMI	YES	NO	NO	NO	NO	NO	IMPROVED
86	SEKH MOHMAD	341	50	M	CP	YES	NO	SM	395	200	EAWMI	YES	1	NO	NO	YES	NO	IMPROVED
87	FERNANDAS	344	50	M	CP VOM	YES	YES	SM /ALC	610	194	ASMI	YES	NO	NO	VT	YES	NO	DIED
88	BASHEER	346	75	M	CP SW	YES	NO	SM/ALC	380	190	ASMI	YES	NO	LBBB	VPC	YES	NO	DIED

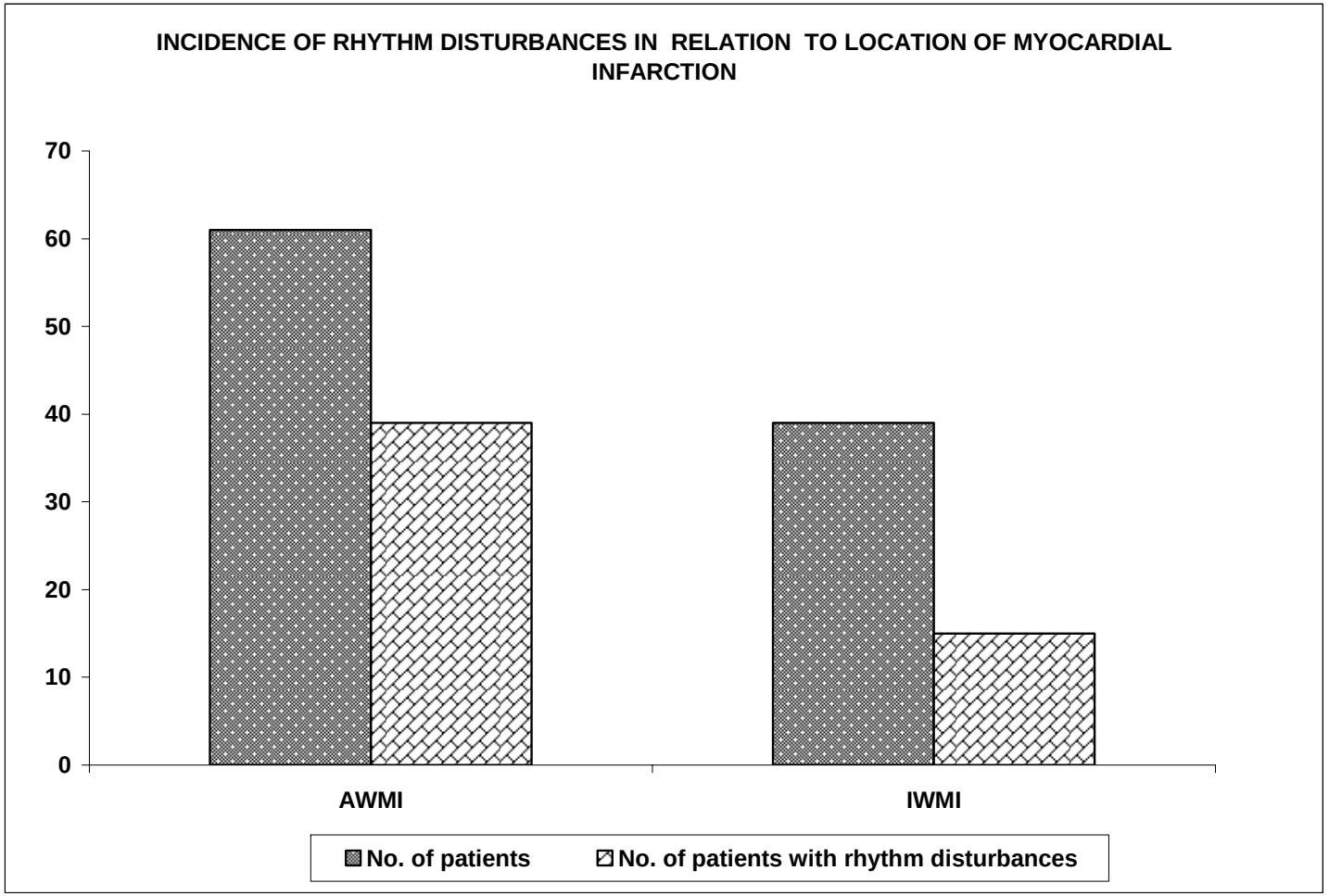
SI NO.	Name	CC NO	Age	Sex	Pres. Comp	DM	HT	Pa.His	CPK	S CHO	ECG	ECHO	LVAV BLOCK	IV CB	OTHERS	RYH	DIS THRO	OUTCOME
89	KALAI SELVAN	349	60	M	CP BR	NO	YES	IHD	720	210	EAWMI	YES	NO	NO	NO	NO	YES	IMPROVED
90	SUMAN	351	37	M	CP	NO	YES	NIL	510	220	ASMI	YES	NO	NO	NO	NO	YES	DIED
91	NANDAGOPAL	356	48	M	CP	YES	NO	SM	378	175	EAWMI	YES	NO	NO	NO	NO	NO	IMPROVED
92	MANIVANAN	358	62	M	CP SW	YES	YES	NIL	456	186	IWMI	YES	NO	NO	NO	NO	NO	IMPROVED
93	NARENDRAN	359	50	M	CP	YES	NO	IHD	478	192	IWMI RVMI	YES	NO	NO	NO	NO	YES	IMPROVED
94	RAJENDRAN	379	68	M	CP	YES	NO	NIL	524	210	ASMI	YES	NO	NO	NO	NO	YES	IMPROVED
95	GANAPATHI	380	47	M	CP	NO	NO	NIL	220	196	ASMI	YES	NO	NO	NO	NO	YES	IMPROVED
96	MALLIGA	382	48	F	CP SYN	YES	YES	SM/ALC	260	125	IWMI	YES	2	RBBB	VPC	YES	YES	IMPROVED
97	KAMESH	385	62	M	PAL SW	YES	NO	SM/ALC	526	280	AWMI	NO	NO	NO	VT	YES	NO	DIED
98	SULAIMAN	386	48	M	CP	YES	NO	NIL	230	240	IWMI	YES	NO	NO	NO	NO	NO	IMPROVED
99	ANWAR	388	40	M	CP SW	YES	NO	SM/ALC	512	241	ASMI	YES	NO	**NO**	NO	NO	YES	IMPROVED
100	NAGARAJ	400	50	M	CP	YES	NO	NIL	512	220	IWMI RVMI	YES	NO	NO	NO	NO	YES	IMPROVED

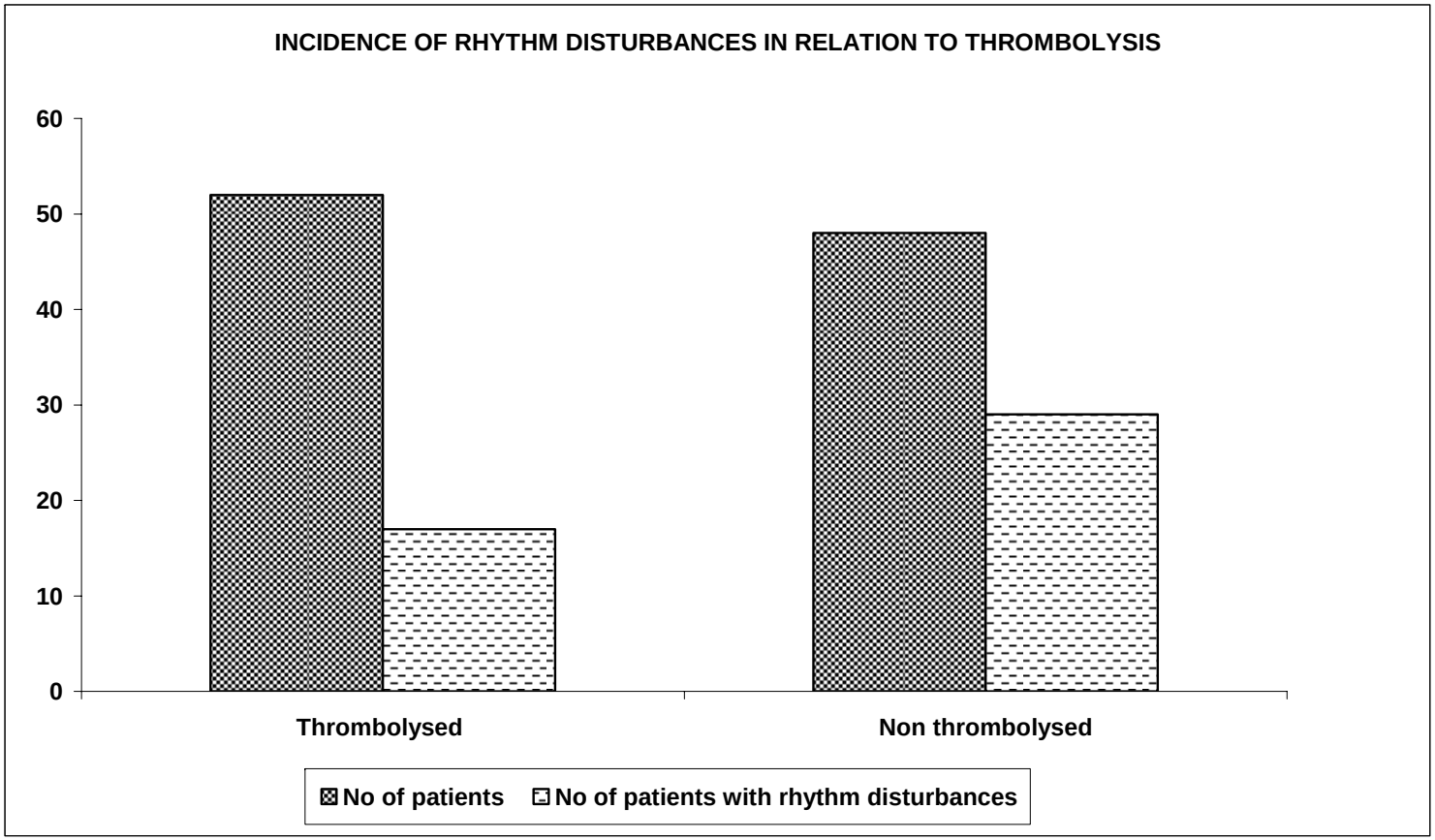

INCIDENCE OF RHYTHM DISTURBANCES IN PATIENTS WITH ACUTE MI
46%
54%
No of patients with rhythm disturbances
No of patients without rhythm disturbances

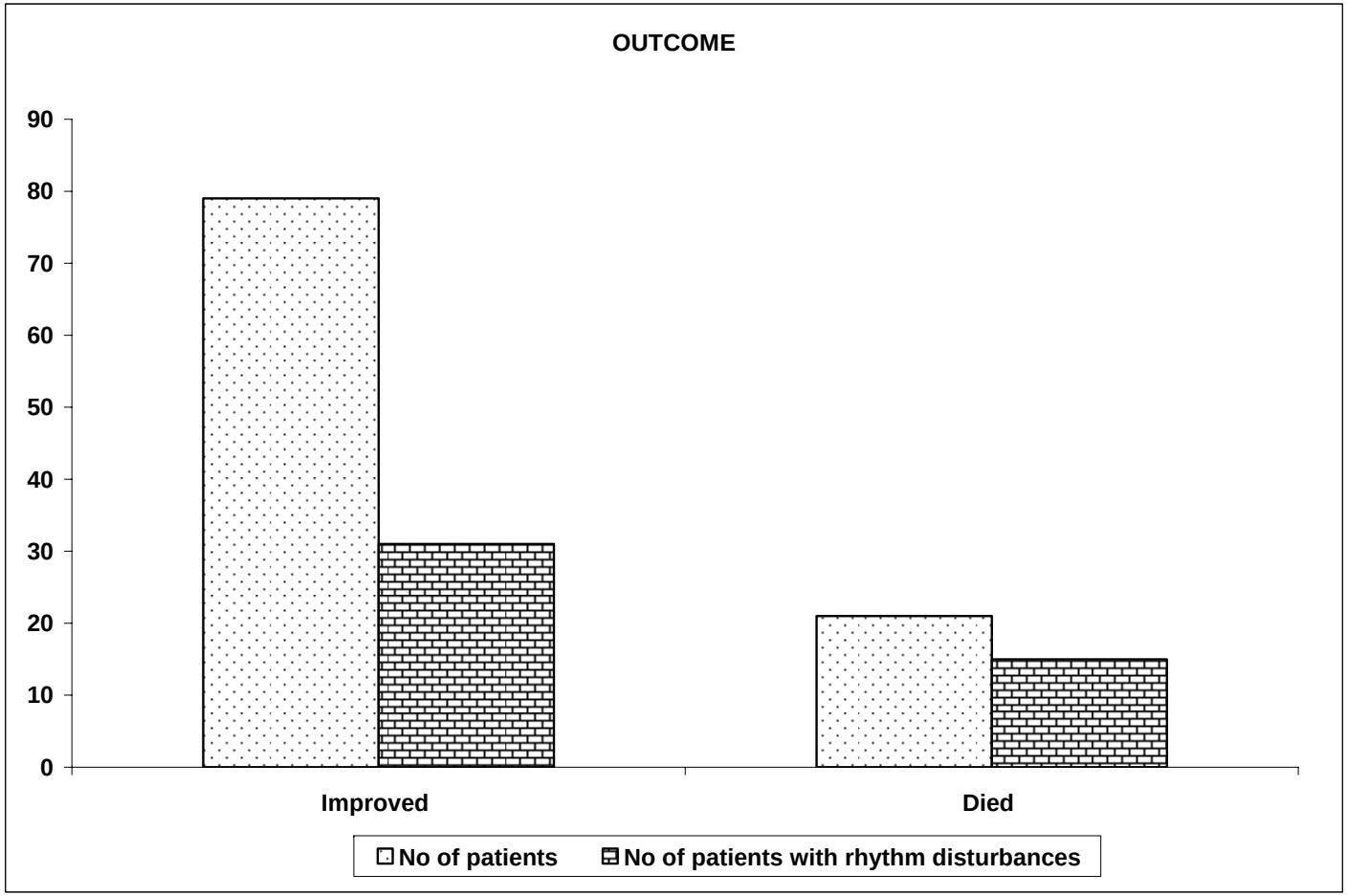

INCIDENCE OF RHYTHM DISTURBANCES IN PATIENTS WITH ACUTE MI ACCORDING TO AGE
35
30
25
20
15
10
5
0
20-29 Years
30-39 years
40-49 years
50-59 years
60-69 years
70-79 years
80-89 years
Number of Patients
No. of conduction defects


INCIDENCE OF RHYTHM DISTURBANCES IN PATIENTS WITH ACUTE MI ACCORDING TO SEX
90
80
70
60
50
40
30
20
10
0
Male
Female
Total -100
Ryhthm Disturbance


INCIDENCE OF RHYTHM DISTURBANCES IN PATIENTS WITH VARIOUS RISK FACTORS IN ACUTE MI
60
50
40
30
20
10
0
Smoking
Diabetes
Hypertension
Alcohol
Hypercholesterolemia
No. of Patients
Patients with Rhythm Disturbance


INCIDENCE OF RHYTHM DISTURBANCES IN RELATION TO LOCATION OF MYOCARDIAL INFARCTION
70
60
50
40
30
20
10
0
AWMI
IWMI
No. of patients
No. of patients with rhythm disturbances


INCIDENCE OF RHYTHM DISTURBANCES IN RELATION TO THROMBOLYSIS
60
50
40
30
20
10
0
Thrombolysed
Non thrombolysed
No of patients
No of patients with rhythm disturbances


OUTCOME
90
80
70
60
50
40
30
20
10
0
Improved
Died
No of patients
No of patients with rhythm disturbances